myrobalan and the Marianna plums. Selection number 29 of the myrobalan plum and 2624 of the Marianna have proved highly resistant and are good stocks for commercial varieties of plums and apricots. These stocks, especially Marianna 2624, also are resistant to armillaria root rot and to the effects of waterlogging of heavy soils. Replanting affected spots with Marianna 2624 rootstock on which plum or apricot can be grown offers the best promise in situations to which these fruits are adapted.

MANY CHEMICALS have been tested against this fungus in closed cans of soil and several have been tried in orchards. To date no encouragement has come out of these tests.

Every possible precaution should be taken to prevent the spread of the fungus during irrigation or other cultural operations as well as in the movement of nursery stock.

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*For further reading the authors suggest Harold E. Thomas'* Studies on Armillaria mellea (Vahl) Quel., Infection, Parasitism and Host Resistance, *Journal of Agricultural Research, volume 48, pages 187–218, 1934, and* Identification and Treatment of Armillaria Root Rot, *Almond Facts, March 1942.*

# Coryneum Blight of Stone Fruits

*E. E. Wilson*

Coryneum blight also is called shot hole, corynosis, a peach blight, fruit spot, winter blight, and pustular spot. It is caused by the fungus *Coryneum beijerinckii* (*Clasterosporium carpophilum*), which was first noted in France in 1853 and subsequently in North and South America, Africa, Australia, and New Zealand. It has been found in Michigan, Ohio, and many other parts of this country, but it is serious only in the far western States.

Its natural hosts belong to the genus *Prunus*. The peach (*P. persica*), apricot (*P. armeniaca*), nectarine (*P. persica* var. *nectarina*), almond (*P. amygdalus*) and sweet cherry (*P. avium*) are the main ones. Other known hosts are the European plum (*P. domestica*), the wild cherries (*P. serotina*, *P. virginiana*, and *P. padus*), the cherry-laurel (*P. laurocerasus*), and *P. davidiana*.

Coryneum blight differs in severity on the various stone fruits. For example, it is seldom found on sweet cherries in California, but it is serious on them in the Pacific Northwest.

It attacks dormant leaf buds and blossom buds, blossoms, leaves, fruits, and twigs. In California, the extent to which these structures are affected varies greatly among the four principal hosts. On peaches and nectarines, for example, twigs and dormant buds are severely affected and so are leaves and blossoms at times, but by and large peach fruit are not seriously affected. But on apricots twig infection is rare,

the disease being confined largely to dormant buds, leaves, and fruit. On almonds, twig lesions occur but seldom abundantly. Leaf infection is common on almond and sometimes blossom infection is common.

The lesions of coryneum blight at first are small, round purplish-black spots on the surface of the affected part. The lesion seldom is more than 5 millimeters in diameter on fruit, but as the fruit develops the surface of the lesion becomes raised and scurfy. On young leaves the diseased areas may expand rapidly and kill large areas of the blade. If the lesions are on the petiole, the leaf is killed outright. Frequently large numbers of young leaf clusters are killed by lesions that develop on the base of the petioles. On the blades of mature leaves, the affected areas soon are separated from the nonaffected tissue by abscission zones and thereupon fall away. Newly formed leaves with only a few lesions will drop, but older leaves commonly remain on the tree despite a number of lesions.

Affected buds are darker in color than unaffected ones. Often, particularly on apricots, they are "varnished" by a thin film of dried gum. The fungus kills the bud by invading apparently between the bud scales and by attacking the twig near the base of the bud. Invasion of the bud is common with all hosts.

On peach and nectarine twigs, the lesions, which at first are small, purplish, raised spots, expand into elongated, necrotic cankers. Many twigs, especially those in the lower part of the tree where much of the best fruit is produced, are killed in late spring and early summer. The disease thereby reduces the bearing surface of the tree for a number of years to come.

The two names of the causal organism reflect the different opinions as to the nature of its fruiting structure. That need not concern us now, but we shall pay close attention to the life cycle of *C. beijerinckii*. Here, too, some disagreement exists regarding the number of fruiting structures. Paul Vuillemin in France reported in 1888 that the fungus has a sexual stage, which he named *Ascospora beijerinckii*. R. Aderhold in Germany, R. E. Smith in California, and Geoffrey Samuel in Australia later studied the fungus carefully, but none found the sexual stage. Apparently, therefore, only one type of propagative structure is regularly produced—a four- to six-celled, ovoid, yellowish conidium, borne on a short stalk (conidiophore), which rises from a simple cushion of fungal cells.

When the conidium drops from the conidiophore to a favorable place, it germinates and produces a germ tube from one or more of its four or five cells. A conidium deposited on a twig or leaf and surrounded by a film of moisture quickly produces a gelatinous sheath about itself. The sheath anchors the conidium to the substrate so that rain does not easily dislodge it. Infection is accomplished by a slender projection from the germ tube, which penetrates the host tissue. On leaves the infection hypha penetrates directly through the cuticle and is seldom if ever found entering stomata. After entry of the infection hypha, the fungus produces mycelium between the walls of the host tissue. From this mycelium loosely packed cushions of hyphal cells then form, emerge to the surface, and give rise to conidia.

*Coryneum beijerinckii* therefore passes its entire cycle on the tree. Contrary to Vuillemin's belief, the mycelium in leaves that fall to the ground apparently does not play a part in its development. For its perpetuation the fungus depends on the mycelium and conidia that remain alive in the diseased buds and twigs. In California the fungus undergoes a period of unfavorable conditions in summer, when lack of rain and probably high temperatures prevent its development. Throughout this period the conidia inside blighted dormant buds retain their viability, although those on the surface of twig lesions do not. Hence conidia are present and readily available for infection even during the inactive stage of the

fungus. After rains begin in autumn, conidia develop on the surface of twig lesions and new conidia develop inside diseased buds. Occasionally the fungus survives in diseased blossoms that remain in the tree.

As I have noted, the incidence of twig and bud infection differs in the four main hosts. Consequently we find that on apricots, which are not subject to twig infection, the diseased dormant buds are the primary inoculum sources; on peaches and nectarines, twigs and buds are equal in importance as inoculum sources; and on almond, blighted spurs are probably more important than blighted buds. Fruit, on the other hand, is not an important source; conidia are seldom produced on fruit lesions.

Winds play a secondary role in disseminating the conidia. Moving air is ineffective in detaching the conidia from the conidiophore; air currents cannot remove the conidia from inside the blighted buds. Water washing over the twigs and buds readily accomplishes this step in dissemination and, in addition, spreads the spores about the tree. Conidia washed downward by rain subject the twigs and buds in the lower part of the tree to much heavier infection than those in the upper part. The dispersal of conidia upward and outward from their sources apparently is accomplished by spattered and wind-blown spore-laden raindrops.

For the infection process no less than for dispersal, the fungus requires the moisture supplied by rain. To germinate and infect the host the conidium must be in a film of water. Consequently no infection occurs during dry weather. Infection is initiated only when the susceptible parts are wet for a long enough time to permit the conidium to germinate and the germ tube to penetrate the host tissue. Once that occurs, the fungus is no longer dependent on the moisture supplied by rain but obtains it from the host. The length of time required for infection, in turn, is influenced by temperature.

Our information on the relation between temperature and growth of the fungus is not extensive, but a few general statements are possible regarding infection and development of the lesions. Although temperatures in California in winter are below the optimum for growth (about 72° F.) of the fungus, probably they are seldom so low as to prevent infection and disease development. It should be remembered in this connection that the temperature is usually higher in rainy than in dry winter weather. In fact, the temperature during rainy periods seldom goes lower than 45° but often as high as 60°. We find therefore that infection occurs during the long rains of midwinter. The influence of temperature is evident, however, in the length of time required for infection and the length of the time between infection and the first visible symptom.

To permit infection at the most favorable temperature, the host parts must remain wet for several hours. Temperature below the optimum prolongs the time necessary for the fungus to gain entrance to the host and lengthens the incubation period. In warm spring weather it is not unusual to find lesions developing 5 to 6 days after the rainy period which initiated infection. In winter 15 to 18 days may elapse before lesions can be seen.

Knowing something about the conditions necessary for infection and having a general knowledge of length of incubation periods, we can obtain considerable information from data such as that presented in the chart on page 709. Here is represented by graphs the increase in the number of twig and leaf lesions during the 1935–36 season and the rainfall record for that season. We first note that rains fell in October, November, and early December and a small wave of twig infection was found on December 24.

On the basis of what I said earlier regarding the length of incubation periods, this infection probably was initiated during one or both of the December rains, the last of which ended 12

days before the lesions first became visible. Apparently, therefore, infection was not initiated during the October and November rains, probably because they were too short to permit it. Data obtained in other years support the view that the first autumn rains not uncommonly are too short to permit twig infection, but those rains often initiate abundant leaf infection, which, however, is of little consequence to the tree because the leaves fall shortly thereafter. Further examination of the chart reveals that large numbers of twig lesions developed at two other times during the winter. Each of the developments followed an extended rainy period.

Here again, data obtained in different years have agreed in all major respects. Consequently we can draw some general conclusions from them. First, twig and dormant bud infection in serious amounts is not likely until the longer rains of winter begin. Second, such infection may occur any time during the winter or early spring when rains of sufficient length do occur. This information has been of great value, as we shall see, in formulating a fungicidal control program for the disease.

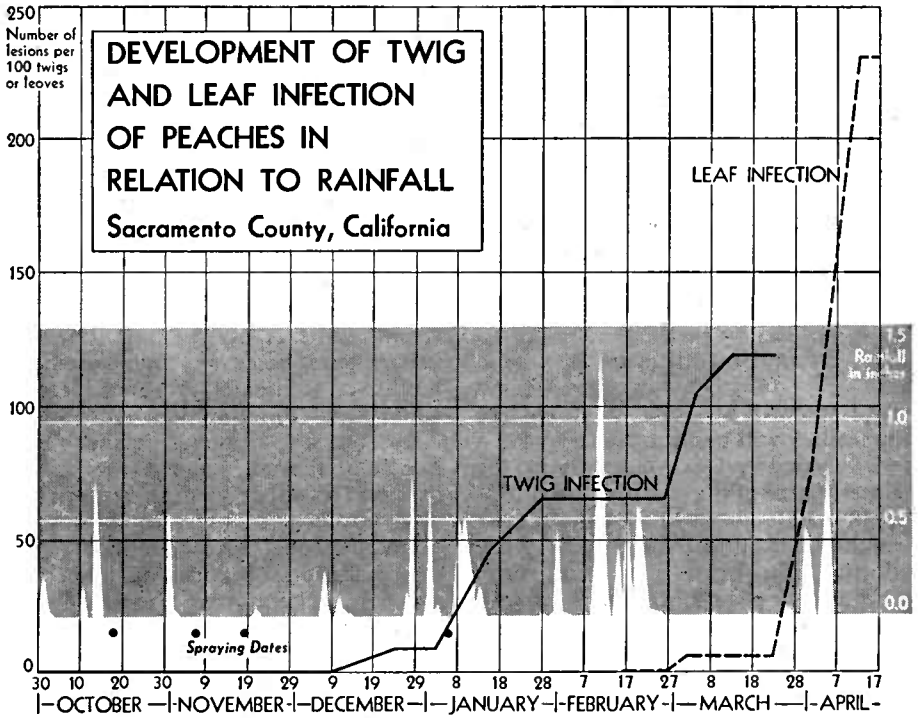
BECAUSE *Coryneum beijerinckii* survives from one season to the next only in the tree, the elimination of the hold-over sources should aid in controlling the disease. Removal of the diseased twigs at pruning time is both impractical and destructive to the fruiting wood.

Eliminating the conidia in and on the diseased parts by means of chemical sprays so far has proved only partly successful. Several phenol and cresol derivatives, notably sodium dinitro-o-cresolate and sodium pentachlorophenate, destroy a great many of the conidia. Applying them as sprays to the dormant tree has noticeably reduced the incidence of the disease at times. Apparently however, they do not destroy the mycelium of *C. beijerinckii* in diseased twigs, so the fungus soon produces a new supply of conidia.

With peaches, control of coryneum blight by means of a protective spray is highly effective. Professor R. E. Smith in 1906 obtained almost complete suppression of new infection from spraying peach trees with bordeaux mixture, 10–10–100. His program, it is safe to say, prevented the abandonment of peach growing in some parts of California. The program consisted in spraying the trees in November or early December and again in February with the bordeaux preparation. The primary purpose of the later spray was the prevention of leaf curl. Omission of this application became possible after better sprayers were developed.

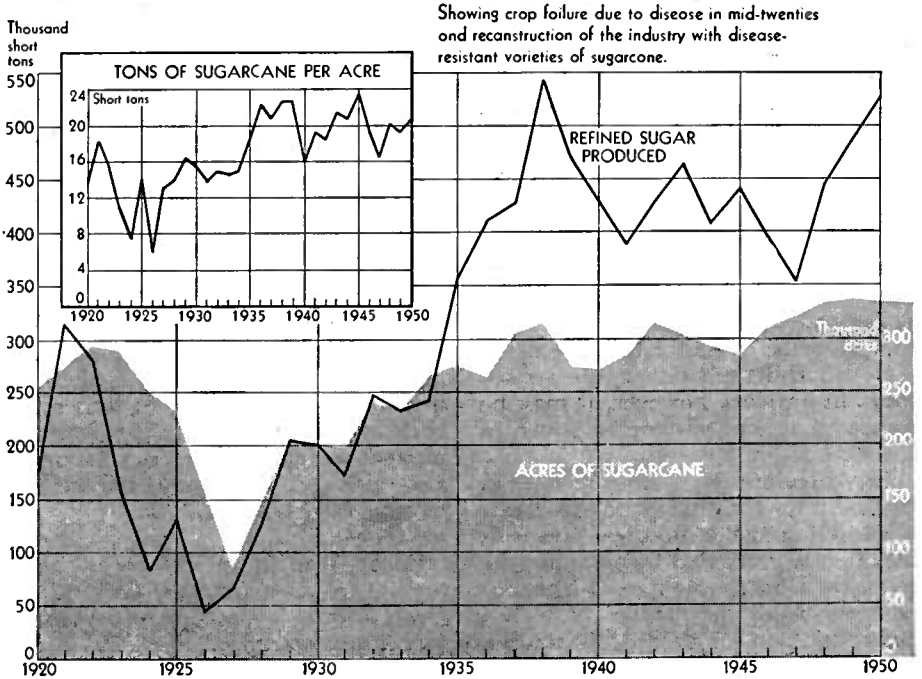
Dr. Smith demonstrated that effective control depended on the timing of the spray application at the beginning of the winter season. He summarized his findings somewhat as follows: Trees sprayed in December were free from coryneum blight and leaf curl; trees sprayed in January were free from leaf curl but affected somewhat by blight; trees sprayed in February and March were free from leaf curl, but severely affected by blight.

In our work at the California Agricultural Experiment Station the timing of the spray treatment in relation to leaf fall was studied closely. The foliage, of course, interferes with deposition of the spray on the twigs. It is desirable therefore to delay that treatment until the leaves are off the trees. Our results indicated that that can be done if the treatment is not delayed too long. The practice of some growers of spraying any time between leaf fall and late December is not to be recommended because severe twig infection may have preceded the treatment. In fact, a small amount of twig infection may occur before leaf fall. Between 1935 and 1941, for example, tests showed that infection before leaf fall occurred in two seasons out of the six, but in only one season was the amount sufficient to cause appreciable damage to the tree. Apparently, therefore, the practice of spraying soon after leaf fall is a safe one.




## CANE SUGAR PRODUCTION IN THE UNITED STATES 1920–1950

Showing crop failure due to disease in mid-twenties and reconstruction of the industry with disease-resistant varieties of sugarcane.



The single autumn treatment is expected to prevent twig infection throughout the dormant season, when heavy and prolonged rains occur. To be effective over such a long period, the fungicide deposit must have superior weather-resistant qualities. Our experience is that bordeaux mixture meets the requirements admirably—it is toxic to the conidia of *Coryneum beijerinckii* and it has excellent weather resistance as well. One-fourth or more of the copper deposited by bordeaux in autumn often is found on the twigs in spring. Few other fungicides can meet those requirements. A single treatment of lime-sulfur will protect the twigs for a short period but not throughout the winter. Some of the so-called fixed copper fungicides prove satisfactory if their weather resistance is increased by the addition of a sticker. Petroleum spray oil is used for the purpose. To obtain the maximum sticking qualities from such a supplement, the fungicide particles have to be incorporated in the oil phase of the emulsion. A stable oil-in-water emulsion is unsuitable, because the fungicide particles remain in the water phase and are wetted by the oil only after the water has evaporated from the spray deposit on the host. By selecting the proper emulsifier, however, conditions can be made that promote differential wetting of the fungicide particles by the oil.

We have considered so far the control of coryneum blight in peaches only. The procedure for controlling the disease in apricots is similar in that an autumn spray is applied. Its primary object is to prevent the fungus from attacking the dormant buds. Because leaf and fruit infection cause serious losses in apricots, however, it may be necessary to apply another spray in the spring.

Investigations in California and in Australia on the timing of this treatment demonstrated that leaf and fruit infection is prevented by spraying the trees just after the unopened blossoms emerged from the winter buds but before the petals unfolded. At times a second spray soon after petal fall may be necessary but, by and large, the autumn treatment followed by the preblossom treatment is sufficient. As a preblossom treatment is given for the brown rot blossom blight disease in many parts of California, no additional expense is involved in following the two-treatment program.

Early attempts to control coryneum blight in almonds, following the same procedure as for peach, gave indifferent results. It soon became apparent in our tests, however, that coryneum blight on almonds is much less a wintertime disease than suspected. That is to say, twig and dormant bud infection, though present, are not so common as either to destroy appreciable numbers of twigs or to supply abundant conidia for infection. Then, too, infection of dormant buds apparently occurs more frequently in early spring than in winter. In any event, a bordeaux spray treatment just before the blossom buds begin to open materially reduces the number of buds infected. Moreover, such a treatment subsequently reduces the amount of flower and leaf-cluster blighting which can be a serious phase of coryneum blight on almonds. Experience indicated the need for an additional spray to prevent leaf infection in years when rainy weather extended into late spring. This spray is now applied soon after the trees are through blossoming.

The timing of the first treatment was later reexamined and it was decided to forego control of dormant bud infection in favor of more effective prevention of leaf-cluster and flower infection during the blossoming season. The first spray now is applied just after the blossom buds emerge from the winter buds but before petals unfold. A treatment at this stage of host development also aids in preventing brown rot blossom blight disease.

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